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ABSTRACT

This paper is a clinical discussion of post-traumatic stress disorder and violence, particularly as it applies to the Vietnam Post-Traumatic Stress Syndrome. In the first section, the syndrome is described as the sudden onset of explosive rage and unprovoked violence with little or no warning, accompanied by a drastic change in personality. It is noted that, when substance abuse is involved, it is believed that the drug tends to lower the epileptic threshold thereby exacerbating both the onset and intensity of unprovoked rage. The organic aggressive syndrome is described as being associated with certain types of brain lesions, and as possibly originating from prenatal, neonatal, or postnatal factors as well as from brain injury sustained later in life. The diagnostic criteria for the Organic Personality Syndrome are outlined in detail. In the second section, the specific characteristics of the Vietnam Post-Traumatic Stress Syndrome are described. It is stated that, while Vietnam had fewer recorded incidents of acute combat stress than World War II or Korea, it has a far greater legacy of delayed and chronic disorders after the soldiers have returned to civilian life. As victims of their clinical and diagnostic tools clinicians have overlooked the full scope of the Vietnam Post-Traumatic Stress Syndrome. References are included. (TE)

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POST-TRAUMATIC STRESS DISORDER AND VIOLENCE

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I. Understanding Impulsive Aggression:

The recent advent of medical technology, notably computed tomography (CT scan), magnetic resonance imaging (MRI), and radioisotope imaging (PET/scan) allows for significant advances in neuropsychology today. But even prior to the development of the CT scan in 1973 scholars drew attention to the problem of impulsive aggression among children and youth -- especially those with epilepsy (Gunberg & Pond, 1957; Nuffield, 1961; Pollack, 1967; Ounsted, 1969). Other early areas of study included violence and epilepsy among adult criminal offenders (Fenton & Udwin, 1963; Gunn & Bonn, 1971; Rodin, 1973; Klingman & Goldberg, 1975; Lewis, 1975) and violence secondary to drug intoxication (LSD, alcohol, narcotics, amphetamines)(Valzell, 1967; Ellinwood, 1971; Baden, 1972; Sadow, 1973).

Interestingly, the organic aggressive syndrome dates back to 1899, four years after Roentgen discovered X-rays. Later termed the "episodic dyscontrol syndrome" by Karl Menninger, the organic aggressive syndrome is classified as "organic personality disorder - explosive type" in the DSM-III-R. Other major clinical syndromes associated with outbursts of aggression or rage include substance abuse intoxication (alcohol, cocaine, inhalant, sedative, hypnotic or anxiolytic intoxication), intermittent explosive disorders, post-traumatic stress disorders and primary degenerative dementia. Impulsive aggression is also seen as a feature of organic affective disorders (organic mood syndrome - manic state)(Spitzer & Williams, 1980; 1987; Elliot, 1987).

The major feature of these impulsive aggressive disorders is the sudden onset of violence with little or no warning accompanied by a drastic change in personality (affect). The violence itself is often primitive, brutal, sudden and with considerable accuracy (on target). If there are any antecedents (school or work demands, being corrected or mildly reproached...) the resulting physical aggression is greatly disproportionate to these causal factors.

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The single distinguishing feature of the organic aggressive syndrome is a seemingly unprovoked explosive rage. This departs from reactive aggression -- that clearly precipitated by antagonistic factors (Tardiff, 1982; 1984; French, 1985-86). And when substance abuse is involved in the rage it is believed that the drug, and possibly other agents such as aspartame, tend to lower the epileptic threshold thereby exacerbating both the onset and intensity of unprovoked rage (Langevin, et. al., 1987). Moreover, the organic aggressive syndrome is associated with certain types of brain lesions -- notably those located in the frontal lobe, temporal (sides) lobes and the occipital area (back and base of the brain). The etiology of aggressive epilepsy can be due to prenatal, neonatal, or postnatal factors as well as to brain injury sustained later in life (injuries, wounds, medical interventions such as lobotomies). In latent epilepsy among children and youth environmental stressors, including school demands, can exacerbate the onset of seizure activity and subsequent aggressive outbursts (Elliot, 1976).

Exacting the location and type of organic brain damage is a difficult clinical task even with the recent advent of computed tomography, magnetic resonance, and radioisotope imaging. In addition to lesions, infarctions and hemotomas to the brain, a host of skull injuries can also contribute to craniocerebral trauma. Included here are linear, diastatic, comminuted, depressed compound, and blow-out fractures and cysts secondary to skull fractures (Ramsey & Scheer-Williams, 1987).

Biochemical explanations for the organic aggressive syndrome focus primarily upon the role neurotransmitters play in the biochemical processes within the brain. Inferring from animal research, it appears that acetylcholine (the first known neurotransmitter - discovered in the 1920s) contributes to increased aggression due to its link with the autonomic (involuntary) nervous system synapses. Eichelman (1987) sees the relationship between aggression and acetylcholine as an enhancement of central cholinergic activity. Dopamine, the neurotransmitter discovered in 1958 and associated with schizophrenia, also appears to influence aggressive behaviors through its transmission of catechalomine. Psychomotor stimulants, such as amphetamine, methylphenidate (a mild psychomotor stimulant), and cocaine are potential releasers of dopamine and, in high dosages, can induce a paranoid psychosis, as can dopamine drugs (L-Dopa)(Kaplan & Sadock, 1985).

On the other hand, GABA (Gamma-Amino-Butyric Acid) and serotonin are two neurotransmitters whose function seems to be to reduce aggression.

Some studies claim that therapeutic dosages of denzodiazepines have a calming effect of aggressive behaviors through its enhancement of central GABAergic activity, while other studies indicate that lithium is effective against aggression because of its apparent ability to increase the uptake of serotonin precursor, tryptophan, in the brain (Eichelman, 1987).

As stated earlier when speaking of aggression, we must also take into consideration two major functional disorders whose clinical features mimic the organic aggressive syndrome. These are the post-traumatic stress disorder (PTSD) and alcohol and drug intoxication (the clinical category of "psychoactive substance-induced organic mental disorders actually falls within the area of organic syndromes). Either clinical disorder, in itself, can produce the same sudden, intense aggressive reaction greatly disproportionate to real world stressors. Indeed, clinicians treating violent (aggressive/suicidal) adolescents for substance abuse are finding that a substantial number of these individuals were the victim of child abuse (sexual, physical or psychological trauma as a child) and that drinking and/or drug abuse are often merely an associated feature. Substance abuse is seen as a form of self-medication in an attempt to cope with the anxiety surrounding the earlier child abuse -- much of which was suppressed (a post-traumatic reaction).

Again, research with lower primates indicates that trauma, such as that experienced by humans suffering from the post-traumatic stress disorder, can alter the neurophysiology of neurotransmitters in the brain -- notably dopamine and norepinephrine. Together, these neurotransmitters increase the sensitivity of the neuroreceptors (accounting for the disproportionate paranoid delusion associated with minor stressors) as well as initiating the "flight or fight" automatic mode of the Central Nervous System (this accounts for the impulsiveness and intensity of the aggression). The current thinking is that alcohol, amphetamines, and cocaine induce similar neurophysiological changes involving these neurotransmitters. The difference, however, between the changes associated with drug intoxication and PTSD and that linked with the organic aggressive syndrome is that there is some evidence to indicate an eventual reversal (normal stabilization within the brain chemistry) regarding substance intoxication and PTSD following successful treatment. The promise for such success with people afflicted with the organic aggressive syndrome is not as promising although some hope hinges upon the use of beta blockers and in the use of surgical interventions (such as severing the corpus collusum in an attempt to prevent interictal transmission of epileptic seizures during acute attacks)(Elliot, 1987; Franzen & Lovell, 1987; Green &

Costain, 1981; Hartman, 1987; Langevin et. al., 1987; Nace, 1987; Roth, 1985; Silver & Yudofsky, 1987; Stewart et. al., 1987; Yudofsky et. al., 1987).

The DSM-III-R lists the following criteria for the Organic Personality Syndrome.

A. A persistent personality disturbance, either lifelong or representing a change or accentuation of a previously characteristic trait, involving at least one of the following:
    1. affective instability, e.g., marked shifts from normal mood to depression, irritability, or anxiety.
    2. recurrent outbursts of aggression or rage that are grossly out of proportion to any precipitating psychosocial stressors.
    3. markedly impaired social judgement, e.g., sexual indiscretions.
    4. marked apathy and indifference.
    5. suspiciousness or paranoid ideation.
B. There is evidence from the history, physical examination, or laboratory tests of a specific organic factor(s) judged to be etiologically related to the disturbance.
C. This diagnosis is not given to a child or adolescent if the clinical picture is limited to the features that characterize Attention-deficit Hyperactivity Disorder (The Attention-deficit Hyperactivity Disorder does not include sudden and recurrent outbursts of aggression or rage. Initiating physical fights, however, is No. 8 in the descending list of 13 characteristics associated with Conduct Disorders for your 18 or younger. Again, this feature should not be confused with sudden and seemingly unprovoked outbursts of aggression or rage).

The Intermittent Explosive Disorder and Intoxication with Psychoactive Substances also fit within the Organic Aggressive Syndrome.

The diagnostic criteria for the Post-traumatic Stress Disorder are:

A. The person experienced an event that is outside the range of usual human experience and that would be markedly distressing to almost anyone (rape or assault; military combat; natural or accidental disasters; torture...).
B. The traumatic event is persistently reexperienced in at least one of the following ways:
    1. recurrent and intrusive distressing recollections of the event (in young children, repetitive play in which themes or aspects of the trauma are expressed).
    2. recurrent distressing dreams of the event.
    3. sudden acting or felling as if the traumatic event were recurring (flashbacks, illusions, hallucinations...).

4. intense psychological distress at exposure to events that symbolize or resemble an aspect of the traumatic event, including anniversaries of the trauma.

C. Persistent avoidance of stimuli associated with the trauma or numbing of general responsiveness (not present before the trauma), as indicated by at least three of the following:
   1. efforts to avoid thoughts or feelings associated with the trauma.
   2. efforts to avoid activities or situations that arouse recollections of the trauma.
   3. inability to recall an important aspect of the trauma (psychological amnesia).
   4. markedly diminished interest insignificant activities (in young children, loss of recently acquired developmental skills such as toilet training or language skills).
   5. feelings of detachment or estrangement from others.
   6. restrictive range of affect, e.g., does not expect to have a career, marriage, or children, or a long life.

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by at least two of the following:
   1. difficulty falling or staying asleep.
   2. irritability or outbursts of anger.
   3. difficulty concentrating.
   4. hypervigilence.
   5. exaggerated startle response.
   6. physiologic reactivity upon exposure to events that symbolize or resemble an aspect of the traumatic event (e.g., a woman who was raped in an elevator breaks out in a sweat when entering any elevator)(Spitzner & Williams, 1987).

II. Attributes of the Vietnam Post-Traumatic Stress Syndrome:

Two major factors distinguish Vietnam combat stress from that which is associated with earlier encounters; World War I (shell shock), World War II (combat fatigue), Korean Conflict (zombie reaction): a basic lack of an esprit de corps (a needed requisite for nourishing the warrior image) among servicemen stationed in Vietnam, and the war's unpopularity at home. These and other factors served to cloud the symptoms surrounding the Vietnam post-traumatic stress syndrome and consequently contributed to therapeutic delays. Some feel that this failure to recognize and treat Vietnam-related post-traumatic stress disorders may well have exacerbated clinical syndromes as well as

corresponding undesirable behavior including self- and other-aggression. The extent of these alleged damages will never be known. Leventman addressed this issue on how clinicians may have unwittingly denied Vietnam veterans needed therapeutic services: "Indeed, psychologists and psychiatrists may themselves become victims of their clinical and diagnostic tools. They may so predispose them to see only symptoms of psychopathology that the veterans they treat tend to become just another collection of sick types ultimately separated from the conditions that produce them" (1978: 294).

The implication here is that the American clinical community, by and large, contributed to the negative stereotypical image associated with Vietnam and those who participated in this conflict. A serious clinical bias, perhaps the most significant subjective factor, is that of ethnocentrism. A profile of "troubled" Vietnam veterans, those most likely to suffer from post-traumatic stress disorders, are from "disadvantaged" and/or "minority" backgrounds, or both. Moreover, they are persons who most likely have experienced failures in education, jobs and life in general. Alcoholism, drug abuse and less than honorable discharges fill out the profile - clearly the antithesis of the college-educated clinicians (most who perhaps did not see service in Vietnam)(Zarcone, 1977).

This class distance and its corresponding inferential bias leads to the "fatal flaw" fallacy, posited Leventman (1978). That is, clinicians tend to dismiss veterans' claims of a Vietnam connection. Alternatively, they commonly offer a clinical picture which attributes the cause of these "troubles" to antisocial behavior which they see as preceding the patient's Vietnam tour and continuing after, but not being directly related to this duty. Unfortunately, the military and Veterans Administration contributed to this image. Widespread drug escapism, expeditious, and often misleading, administrative discharges, coupled with individual, as against unit, replacements and relatively short tours (10-13 months) all contributed to the unusual Vietnam phenomenon - a low recorded incident of combat stress. Unfortunately, these same factors seem to be the root cause of the delayed stress which afflicts a disproportionate number of Vietnam veterans.

Commenting on this phenomenon, the Presidential Task Force on Mental Health Problems of Vietnam Era Veterans noted: "Thus the battlefield in Vietnam may well have produced fewer immediate psychiatric casualties requiring evacuation and discharge than did earlier conflicts, but it left a far greater legacy of delayed and chronic disorders which

arise and persist long after the soldier has returned to civilian life" (PCMH, 1978: 1333). Here the Task Force distinguishes between "acute combat reactions," those traditionally associated with combat stress, and "delayed combat reactions." While the symptoms of both these combat stress reactions are similar - the latter is much more difficult to identify, report, and treat. Another dimension of this dilemma is that many of the emotional disturbances which seem to make clinical sense within a combat situation are often displaced overtime and manifest themselves in seemingly inappropriate civilian situations years following military separation.

The Task Force lists the major stress reactions stating that : "the more delayed and diffuse the psychiatric manifestations, the less likely it is that their connection with Vietnam service will be recognized" (PCMH, 1978: 1333). The major stress reactions mentioned in the report are: "affecting muting," that behavior characterized by detachment, withdrawal and the diminished capacity to feel; "guilt," that behavior characterized by a sense of failure to see more combat, or be more effective in combat, or to win the War; "other disturbances," most notably obsessions with death, hyperalertness, startle reactions, sleep disturbances, nightmares, rage, aggression, mistrust, paranoia, and depression. These symptoms are often further complicated by drug abuse and alcoholism, stigmatization (societal reaction to the Vietnam conflict and Vietnam veterans), family breakdown, and a failure for these veterans to integrate back into society.

Another significant factor distinguishing "acute combat reactions" from "delayed combat reactions" among Vietnam veterans is that most of the delayed reactions afflict veterans attached to "support" as against "combat" units (Panzarella, 1978). Clearly, this situation merely contributes to the fatal flaw fallacy to which Leventman (1978) alluded. Understandably, a clinician not exposed to the stresses of Vietnam, stressors which tended to permeate all sectors of Vietnam, might conclude that veterans without direct combat experience might be malingering or perhaps overstating their cases.

Indeed, the easiest delayed stress disorders to recognize are those which represent the exception to the norm - those which are related directly to combat situations. Apostle illustrated such a case in the Bulletin of the American Academy of Psychiatry and the Law. Here he argued that latent combat stressors can surface "unconsciously" years later, even in civilian life, especially if they are triggered by additional, more contemporary, stressors: "A Vietnam warrior is presented as

having a post-traumatic stress disorder in which, under a situation of acute stress (the loss of his children), he reexperienced feelings and behavior similar to those he had experienced in Vietnam; during this time, he was not consciously aware of his actions (aggravated assault)" (1980: 430). Since then, more Vietnam veterans have successfully used this legal defense as a justification for violent outbursts - including homicide (Press, 1981).

Paradoxically, while the "Unconsciousness defense" represents a combined clinical and legal effort to better understand the Vietnam post-traumatic stress syndrome, it, by focusing on those exceptional cases of delayed acute combat stress, alternatively, and perhaps unwittingly, fuels the fatal flaw fallacy by seemingly restricting these "recognized" delayed stress situations to only combat veterans. The few psychiatric assessments of the Vietnam delayed stress syndrome tend to lend credence to this view (Borne, 1970; Shatan, 1978). The symptoms associated with Vietnam post-traumatic stress disorders are generally qualified to include direct combat experience:

1. Guilt feeling and self-punishment (about those killed and wounded on both sides).
2. Feeling scapegoated (blaming the victims, notably the veterans themselves).
3. Rage and other violent impulses against indiscriminate targets (expression of feelings of betrayal and manipulation...).
4. Combat brutalization and its attendant "psychic numbing" (product of military process of enemy dehumanization, e.g., referring to the enemy as "slopes," gooks," "dinks,"...).
5. Alienation from one's own feelings and from other people (a result of combat defenses needed for survival during the Vietnam tour...).
6. Doubt about continued ability to love and trust others (the internalization of the alienation mentioned in #5 above).
7. "Impacted Grief" (inability of veterans to purge their memories of Vietnam service and the unwillingness of the American public to recognize and accept these veterans' "call to duty").

There is little doubt that these symptoms represent a valid and comprehensive clinical assessment of Vietnam post-traumatic stress disorders. It is imperative, however, that clinicians also realize that most, if not all, of these symptoms can also be associated with "support" veterans stationed in Vietnam, especially in light of the pervasive terror and potential for violence throughout Vietnam during that period.

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